Fighting Back

IT IS GOOD to see physicians who have been the victims of unjustified and unjustifiable law suits beginning to fight back. They are suing lawyers or others who sued them or who included them in malpractice suits when there was no legitimate basis for claiming the physicians contributed in any way to the injury the patient alleged. And there has been some success. A pathologist in southern California was awarded a $10,000 settlement of a countersuit for malicious prosecution —where his only involvement in the case was that he did the autopsy on the deceased patient. The attorney had refused to dismiss the action against the pathologist until some seven years after the alleged injury occurred—an injury for which the pathologist could not possibly have had any responsibility.

Several state and county medical associations are helping their members in counterattacks against attorneys in selected cases when there have been frivolous or malicious suits against physicians. The California Medical Association, by an action of its House of Delegates in February, undertook to help physicians in some of these cases with legal research and other support when warranted. In Illinois, legal assistance in selected cases is available from the Illinois State Medical Society to the attorney of a member physician who wishes to file a countersuit. The Iowa Medical Society is in the process of developing a similar program, and there are county societies in Florida, Georgia and Texas that are providing such assistance.

It is becoming increasingly evident that physicians and the medical profession are going to have to fight many of their battles in the courts, not only in their own behalf, but in the interests of their patients and the public as well. (The California Medical Association itself has gone to court in the past—and won—in behalf of patients and the public.) This is working within the American system, and medicine has as much at stake as any other segment of our society in making the American system work. Plaintiffs in malpractice lawsuits and their attorneys who have for so long demanded that their claims be submitted to and resolved by a jury may now expect to find their own conduct subjected to jury review.

—MSMW

Lung Abscess

THE CASE PRESENTED in the Medical Staff Conference on lung abscess in this issue is of distinct interest. Unique features include hilar adenopathy and resistance to therapy that is ordinarily effective. Differential diagnosis would include fungus infection, tuberculosis and tumor. Presence of an aspirated foreign body must always be considered, as well, in any case of lung abscess. Appropriate workup ruled out these possibilities. Moreover, there was evidence to indicate that the process was an anaerobic infection and mediastinal adenopathy may be seen with this type of infection on occasion. The fetid odor to the sputum is a definitive clue to involvement of anaerobic bacteria in the infection, provided necrotizing gingivitis or other intraoral anaerobic infection which might impart such an odor to coughed sputum can be ruled out. When the latter type of infection is present, foul or putrid odor of material obtained by transtracheal aspiration establishes the role of anaerobes in the lung abscess. The absence of aerobic bacteria on culture would indicate that the infection was purely anaerobic. As noted by Dr. Murray in the staff conference, absence of a fetid odor does not exclude the possibility of an anaerobic infection since some anaerobes do not produce the offensive volatile products accounting for the odor and since some pulmonary lesions do not communicate with a bronchus.

Other features suggesting anaerobic infection, as summarized by Dr. Murray, include aspiration, the prolonged course of the illness, tissue necrosis, and dental or gingival disease. Although there was no history of unconsciousness or aspiration, the likelihood of aspiration is great in a person with disorientation and catatonic behavior in whom a lung abscess develops in a dependent pulmonary segment (the apical-posterior segment of the left upper lobe). Although anaerobic pulmonary infection may be acute and fulminating, most of such infections run a subacute or chronic course. This is relatively unique among bacterial pulmonary infections. Tissue necrosis may result in lung abscess, usually solitary, as in the patient discussed, or may be in the form of multiple, small excavations (necrotizing pneumonia) in which case the prognosis is much poorer.

Periodontal disease, gingivitis and other dental conditions involving anaerobes are important

background factors for anaerobic pulmonary infection. Unfortunately, many physicians are not familiar enough with dental pathology and describe abnormalities of various types in very general terms (such as "poor dental hygiene" or "poor dental repair") which are meaningless. Anaerobic bacteria do not play a role in the usual type of caries seen in children, but anaerobes may be important in root, pit or fissure caries and in acute necrotizing ulcerative gingivitis, the various stages and forms of periodontal disease, root canal infection and periapical abscess. Additional clues indicating possible involvement of anaerobes include the unique morphology of certain anaerobes on Gram stain of transtracheal aspirate or empyema fluid and failure to obtain likely pathogens on conventional culture of appropriate specimens.

In a prospective study of 26 cases of primary lung abscess,[1] anaerobic bacteria were recovered in 24 (92 percent) and in two thirds of these patients, anaerobes were the exclusive isolates. Thus, lung abscess is primarily an anaerobic infection. The chief anaerobic isolates were Gram-negative anaerobic bacilli (especially Fusobacterium nucleatum and Bacteroides melaninogenicus) and anaerobic streptococci (Peptostreptococcus) and cocci (Peptococcus). These organisms, along with microaerophilic streptococci (these belong in the genus Streptococcus but are considered with the true anaerobes since they will frequently not be recovered unless good anaerobic transport and culture techniques are used), are the most commonly encountered anaerobes in pleuropulmonary infections of the various types in which these organisms play a role.

Dr. Murray stressed the fact that Bacteroides fragilis may be found in up to 20 percent of anaerobic pleuropulmonary infections and that this may need to be considered in selecting the antimicrobial therapy to be used. He also noted that in mixed infection one need not necessarily include drugs active against each bacterial isolate. Bartlett[2] used penicillin G successfully in seven patients with mixed infections involving B. fragilis despite resistance of this organism to the agent employed. I prefer to include an agent active against B. fragilis (clindamycin or chloramphenicol) in treating seriously ill patients with suspected anaerobic pulmonary infection until I am certain that this organism is not present or until they are no longer seriously ill.

The failure of the patient presented in the conference to respond to several agents generally active against anaerobes is of interest. The presence of resistant facultative bacteria such as Staphylococcus aureus or Klebsiella pneumoniae seems unlikely as these should be recovered readily on aerobic culture. Since there was no evidence of obstructive pathology or an undrained collection of pus to account for failure of response and since the patient responded rapidly to administration of clindamycin subsequently, the most reasonable explanation is the presence of anaerobes resistant to the agents used earlier.

There is relatively little published information on activity of cephalexin against anaerobes, but unpublished data from our laboratory (D. F. Busch, V. L. Sutter, and S. M. Finegold) indicate relatively good *in vitro* activity against anaerobes susceptible to penicillin G. The patient subsequently received rifampin, along with isonicotinic acid hydrazide (INH®), for possible tuberculosis. Rifampin is very active *in vitro* against most clinically important anaerobes except Fusobacterium varium, F. mortiferum and Clostridium ramosum.[3] However, as with other types of bacteria, resistant mutants occur with significant frequency. There is essentially no clinical experience with rifampin in anaerobic infections, but one would anticipate problems with resistance if the agent was used alone (isonicotinic acid hydrazide has no activity against anaerobes).

Penicillin G was next administered, again with unsatisfactory results—despite a dosage of 10 to 12 million units per day. My own feeling is that it is not feasible to treat many or most Bacteroides fragilis infections with even very high doses of penicillin G, although, as noted in the study by Finegold and Bartlett,[2] administration of penicillin may be effective when B. fragilis is part of a mixed flora. Ten percent of strains of B. fragilis are resistant to even 500 units per ml of this agent (V. L. Sutter and S. M. Finegold, unpublished data). Occasional strains of other anaerobes (such as Bacteroides melaninogenicus and Fusobacterium varium) are also very resistant to penicillin. It should be noted that antibiotic disc susceptibility results obtained by the Kirby-Bauer technique or other techniques not specifically standardized for anaerobes (and the particular type of anaerobe being tested) may be misleading. It should also be noted that, while penicillin G is usually the drug of choice for anaerobic pulmonary infection, other penicillins are not necessarily

suitable. Methicillin, nafcillin and the isoxazolyl penicillins (oxacillin, cloxacillin and dicloxacillin) are distinctly less active than penicillin G. Cephalosporins generally have good activity against anaerobes except for Bacteroides fragilis.

The differential diagnosis of lung abscess is outlined in excellent fashion by Dr. Murray, as is the pathogenesis of the disease. Proper stress was placed on the fact that in hospital-acquired aspiration pneumonia, nosocomial pathogens such as Staphylococcus aureus and a variety of relatively resistant Gram-negative bacilli may be involved, along with anaerobes, and that therapy must be modified accordingly. This may also be true in the case of patients in nursing homes.

Some additional comments on the importance of proper specimen collection and transport in the specific diagnosis of lung abscess and other anaerobic pulmonary infections are in order. These important considerations are the responsibility of the clinician. When empyema or bacteremia complicate anaerobic pulmonary infection, they offer convenient sources of information on the specific cause of the parenchymal process. Empyema is seen in more than a third of cases. Bacteremia, on the other hand, is uncommon in anaerobic pulmonary infection and may often not include all organisms involved in the pulmonary process. Direct lung puncture is more hazardous in adults than transtracheal aspiration, samples only a very small area and provides only a very small volume of specimen. It is my feeling that it is not established that fiberoptic bronchoscopy, even with use of a nasopharyngeal airway and the covering sleeves technique, can provide a specimen free of indigenous upper respiratory tract flora (where anaerobes are prevalent). The use of an oxygen-free tube or vial is the ideal system for specimen transport. If such a tube is stoppered with butyl rubber, anaerobes will survive at least 24 hours. However, facultative forms in a specimen may begin to multiply after a time and crowd out anaerobes (or distort the quantitative relationships). Accordingly, if a delay of more than two hours is necessary before the specimen in a gassed-out tube can be cultured, the tube should be refrigerated to minimize such multiplication.

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